

### Volume 11 Spring 1998 Pages 52-58

# Vascular Disorders of the Upper Extremity

Richard Moore, Jr., M.D. and L. Scott Levin, M.D.

From the Department of Orthopaedic Surgery, Duke University Medical Center, Durham, NC.

Address Correspondence to: Dr. Richard Moore, Department of Orthopaedic Surgery, Duke University Medical Center, Box 3384, Durham, NC 27710.

**Abstract:** Vascular disorders of the upper extremity present a challenging problem to the clinician. The initial presentation can range from acute limb threatening ischemia to chronic insufficiency. The potentially devastating consequences of these disorders necessitate a timely, thoughtful, and thorough approach to their assessment, diagnosis, and management. The purpose of this paper is to provide a review of the etiology, evaluation, and treatment of selected acute and chronic vascular injuries and disorders of the upper extremity.

#### Anatomy

The brachial artery crosses the elbow anterior to the brachialis muscle belly. It is accompanied by the median nerve medially and the biceps tendon laterally. In the cubital fossa it divides into the radial and ulnar arteries.

The ulnar artery descends into the forearm deep to the flexor carpi ulnaris and is accompanied by the ulnar nerve on its medial side. At the wrist these structures enter Guyon's canal between the pisiform and hook of the hamate and pass into the palm. The ulnar artery continues as the superficial palmar arch which courses radially. In the palm, the arch lies superficial to the branches of the median nerve; however, this relationship is reversed in the digits.

The first constant branch of the superficial arch is the proper digital artery to the ulnar side of the small finger. This branch is usually followed by three common digital arteries

that course in the second through fourth interspaces, respectively. The proper digital arteries arise from the common digital arteries and form the major supply to the fingers. These travel distally adjacent to the flexor tendons and dorsal to the digital nerves. The radial and ulnar digital arteries form an arch across the distal phalanx. After giving origin to the common digital arteries, the arch continues radially, where it normally forms an anastomosis with the superficial branch of the radial artery [23].

The radial artery descends in the forearm deep to the flexor carpi radialis. At the wrist, it gives off the superficial palmar branch, which courses into the palm to join the terminal superficial arch. The radial artery proper turns dorsally and radially to enter the anatomic snuffbox. It gives off branches, which form the dorsal radiocarpal, intercarpal, and metacarpal arches. The artery then dives between the two heads of the first dorsal interosseous muscle where it divides to form the princeps pollicis and the deep palmar arch [8]. The deep branch passes ulnarly, where it usually anastomoses with a deep branch of the ulnar artery. The princeps pollicis artery runs between the first dorsal interosseous and adductor pollicis dividing into the radial and ulnar digital arteries of the thumb.

There are multiple perforating arteries between the deep and superficial arches. These collaterals allow the hand to survive after division of the radial or ulnar arteries, however, incomplete arches have been noted in up to 20% of hands [6]. In these individuals, loss of flow from one artery can leave some or all of the hand ischemic. In a very small percentage of individuals, a persistent median artery, which enters the palm through the carpal tunnel, will make a significant contribution to distal perfusion.

The venous drainage of the hand consists of superficial and deep networks. The superficial system is the principle source of drainage. It is formed by longitudinal vessels, which join to form a dorsal network that ultimately empties into the basilic and cephalic veins. The deep system is much smaller in caliber and accompanies the arterial arches.

#### **History and Physical Exam**

#### History

Perhaps the most important component in the evaluation of vascular disorders is the history. A tremendous amount of information is available, if carefully obtained. The patient's age, handedness, and occupation should always be determined. In the setting of acute injury, the mechanism should be identified, along with when and where it occurred. It is also helpful to determine the position of the extremity at the time of injury to identify what structures were within the zone of injury. This localization is especially important in the upper extremity due to the gliding nature of structures and their significant excursions. Furthermore, the volume and character of bleeding at the time of injury should be questioned. A thorough history will allow for anticipation of potential difficulties with repair such as the need for vein grafting due to segmental loss with blast or avulsion injuries.

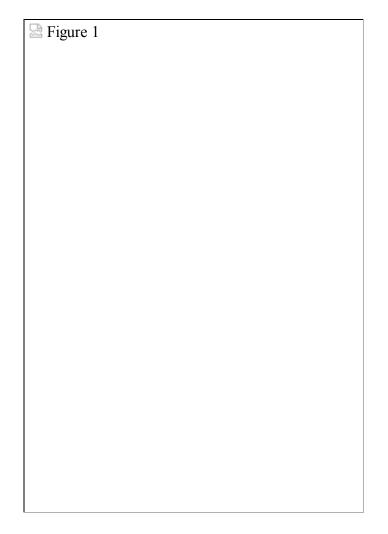
Although arterial lacerations in the setting of acute trauma may present very dramatically, occult injuries associated with penetrating trauma are not uncommon. These injuries must be rapidly and accurately diagnosed to allow appropriate intervention to prevent limb-threatening ischemia. Perry, Thal, and Shires conducted an extensive review of civilian arterial injuries and proposed seven criteria to alert the treating physician to the possibility of underlying arterial injury [32]. Surgical exploration is indicated if any of the following criteria are met: (1) decreased or absent distal pulse; (2) history of persistent arterial bleeding; (3) large or expanding hematoma; (4) major hemorrhage with hypotension; (5) bruit; (6) injury to an anatomically related nerve; and (7) anatomic proximity to a major artery. Additionally, these authors reported an associated major

venous or nerve injury in nearly 40% of cases [32].

Outside the setting of acute trauma, a complete past medical history should be obtained to identify factors that can contribute to vascular disorders. The patient should be questioned concerning systemic disorders such as scleroderma, atherosclerosis, and coagulopathies. Any prior history of injury or surgery in the extremity should be identified as well as any extrinsic causes of vascular insufficiency such as tobacco use, caffeine, or vasoconstrictive drugs.

## **Physical exam**

The physical examination should always begin with a careful inspection of the extremity. Specific note should be made of areas of pallor, hyperemia, cyanosis, and mottling. The presence of scars or ulcerations should be documented (Figure 1).





The heart should be auscultated for irregularity and murmurs. Cardiac dysfunction such as valve incompetence and muscular dysmotility can lead to thrombus formation and embolic events. Similarly, the subclavian system should be auscultated to assess for the presence of a bruit which would indicate a large proximal lesion. Such lesions can have a profound effect on the distal extremity by acting as an embolic source or an obstruction to adequate flow volumes.

All pulses should be palpated beginning with the subclavian and proceeding distally to the axillary, brachial, ulnar, radial at the wrist and in the anatomic snuffbox, and the digital arteries. Bilateral blood pressures should be recorded for the brachial artery. Digital pressures should be obtained to calculate a digital brachial index (DBI). This is calculated by dividing the digital pressure by the brachial pressure [33]. Values < 0.70 are considered abnormal [37]. An obstruction between the brachial and digital vessels will cause the DBI to fall; however, vasospastic disease may not affect the value. Provocative testing may be beneficial in patients with vasospastic syndromes [25].

The relative contributions of the radial and ulnar arteries to the hand can be assessed by the Allen test [2]. The test is conducted by compression of both the radial and ulnar arteries by the examiner. The patient is then asked to open and close their fist several times to exsanguinate the hand. With the hand relaxed, pressure on the radial artery is released while maintaining pressure on the ulnar artery. Both the reperfusion time and distribution of refill in the hand is recorded. This process is then repeated for the ulnar artery and the opposite hand for comparison. Gelberman has reported that the average time to refill in the radial artery is  $2.4 \pm 1.2$  seconds and in the ulnar  $2.3 \pm 1.0$  seconds. He reported that 7% of ulnar and 2% of radial arteries failed to fill the hand in 6 seconds [12]. A delay in filling indicates an incomplete arch and loss of either artery may place a portion of the hand at ischemic risk. The Allen test can also be performed digitally to assess the patency of the radial and ulnar digital vessels.

#### **Diagnostic Modalities**

Hand-held Doppler units provide a simple, accurate, and noninvasive method of evaluating vascular flow. These units function by emitting a low frequency ultrasonic wave that passes through the soft tissues. When the beam encounters a vessel, the frequency is altered by the blood flow and reflected back to the device. The unit gathers the reflected beam converting it into an audible signal. The amount of alteration in the beam's frequency is proportional to the velocity of blood flow; therefore, the quality of the flow within the vessel can be assessed by the quality of the signal. These devices are relatively inexpensive and available in most clinics and hospitals [4].

Diagnostic ultrasound units use high frequency sound waves to produce real time images of vascular structures. High-frequency sound waves are transmitted into and reflected by the soft tissues. Differences in tissue density result in alterations in the reflected signal, which are electronically converted into images. Vascular structures can be accurately visualized and these systems can be used in conjunction with Doppler technology to image flow within the vessels in real time [17].

Plain x-rays are of limited value in assessing vascular disorders; however, associated soft tissue masses, intraosseous lesions, and vascular calcifications can often be visualized. Recent advances in magnetic resonance imaging have led to the development of magnetic resonance angiography (MRA). This technology is relatively expensive and currently not universally available; however, it is non-invasive and produces high resolution images of the vascular structures and surrounding soft tissues. This technique will probably play a larger role in the initial evaluation fvascular problems in the upper extremity in the future [15].

Provocative controlled cooling of the hands with careful monitoring of the finger pulp pressures and temperature has been shown to be a valuable technique in the assessment of circulatory disorders in the upper extremity. Responses have been classified into three types by Koman et al. [24]. **Type I** patients have baseline pulp temperatures of 30 degrees Celsius or greater and fall only 3 to 4 degrees with cooling. During the re-warming period these patients rapidly return to baseline with no pain or discomfort during testing. **Type II** patients are characterized by a baseline temperature below 30

degrees Celsius and fall 8 to 10 degrees during cooling. These patients take significantly longer to re-warm and experience moderate pain with testing. This response indicates an isolated nerve injury or collateralization after vascular injury. **Type III** patients have a baseline temperature of 28.5 degrees Celsius or less and drop 8 to 13 degrees with cooling. These patients re-warm extremely slowly and experience severe pain with testing. The **Type III** pattern is indicative of arterial injury or severe vasospastic disease.

This technique has been combined with other technologies such as plethysmography to provide a more detailed evaluation of vascular disorders [25]. Cold stress testing is also a valuable tool in evaluating recovery and response to treatment.

Arteriography is the gold standard in the evaluation of vascular disorders [15]. The intravascular injection of radiographic contrast agents allows visualization of the vascular anatomy and assessment of their quality and integrity. Although invasive, the technique plays a prominent diagnostic and therapeutic role. The vascular tree of the upper extremity can easily be accessed through the axillary or brachial arteries, but cannulation at this level does not allow visualization of the subclavian system. Proximal lesions can lead to profound vascular changes in the upper extremity and can easily be missed if not studied. The authors recommend that all upper-extremity arteriograms be performed through a femoral puncture to allow adequate visualization of the proximal vessels.

#### **Specific Conditions: Treatment and Outcome**

#### **Acute injuries**

### Intimal injuries

With the development of modern invasive monitoring techniques, intimal injuries caused by arterial cannulation have become relatively common. Intimal trauma from cannulation and indwelling devices can lead to local thrombosis of the vessel, resulting in partial or complete occlusion. This can lead to ischemia or embolization to the smaller caliber distal vasculature.

At the wrist, the radial artery is most commonly involved because of cannulation for intraarterial pressure monitoring. Patients with complete arches usually have sufficient flow through the ulnar system to supply the digits. However, for the 20% of patients with an incomplete arch some or all of the digits may be threatened. Additionally, the thrombosis may act as an embolic source, or the increase in sympathetic tone associated with the lesion can lead to severe vasospasm. An Allen test is mandatory before cannulation of either artery at the wrist.

Some authors have recommended treatment of these lesions with thrombolytics and have reported acceptable results [18]. Others have not been as successful with this approach and have recommended surgical management [20]. The authors of this review recommend prompt surgical exploration and resection of the thrombosed segment to normal intima followed by reconstruction. A preoperative arteriogram is helpful to evaluate the exact location and extent of injury. Primary repair is optimal; however, the resected segment often leaves a gap and reverse interpositional vein grafting is preferable to inadequate resection of injured intima.

#### Lacerations

Over the past two decades advances in microsurgical techniques have led to a dramatic improvements in the treatment and prognosis of arterial lacerations. In the setting of acute trauma, a high index of suspicion for arterial injury should be maintained.

Completely severed arterial stumps will often have surprisingly little bleeding on presentation because of retraction and vasospasm. However, failure to recognize this injury can lead to late bleeding complications. On the other hand, partial arterial lacerations are unable to adequately contract with spasm and will often present with profuse bleeding. This bleeding can be difficult to control with pressure and elevation and usually requires surgical exploration.

The management of an injury to a single vessel with no evidence of ischemia in the hand is controversial [1,9,19]. Isolated unrepaired arterial lacerations in the forearm have been shown by Gelberman et al. to result in consistent alterations in distal perfusion; however, these changes rarely manifest clinically by cold intolerance or ischemia [13]. Additionally, long-term patency rates for repairs of isolated arteries have been reported to be as low as 50% [1,14,30]. Some authors suggest better regeneration of ulnar nerve function following repair at the wrist if the ulnar artery is also repaired [28]; however, others have reported no significant differences [13,19].

Despite the questionable need for repair, the authors recommend exploration of all major arteries near the zone of injury. If significant ischemia is present, then repair of the injured vessel is mandatory; however, if the hand is well perfused, ligation of the arterial stumps is acceptable. Care should be taken to identify and ligate both the proximal and distal stump in order to prevent late complications such as bleeding or aneurysm formation.

When complete, the superficial arch in the palm receives significant contributions from both the radial and ulnar arteries. Therefore, laceration of a complete arch in the palm should not significantly compromise flow to the digits as each stump will be supplied by its contributing artery. Upon exploration, if active bleeding is encountered from both ends of the severed arch, then ligation of the stumps should not place the digits at risk. However, if there is evidence of inadequate flow from one segment, the arch must be repaired or reconstructed.

A significant number of collaterals exist between the radial and ulnar digital vessels in the fingers; therefore, laceration of a single digital vessel is well tolerated and rarely results in symptoms of ischemia. It should be noted that digital artery laceration by a palmar wound is almost always associated with laceration of the accompanying digital nerve because of the volar position of the nerve relative to the artery in the finger.

#### **Chronic Conditions**

#### **Ulnar artery thrombosis**

The superficial position of the ulnar artery at the wrist and its confinement within the relatively unforgiving boundaries of Guyon's canal leave it uniquely subject to injury. Since its initial description by Van Rosen in 1934 [34], thrombosis of the ulnar artery at the wrist attributable to repetitive trauma, or "hypothenar hammer syndrome"[7], has been widely reported. Despite its presence in the medical literature, it often remains unrecognized or misdiagnosed.

Ulnar artery thrombosis most often occurs in males of working age and is associated with occupations or activities which involve repetitive blunt trauma to the hands. The disorder primarily affects the dominant hand and has been related to hammering with the heel of the palm, pneumatic devices, and prolonged pressure over the hypothenar eminence.

The pathogenesis of the disease is believed to stem from intimal damage [26,27]. The intimal disruption leads to activation of the clotting cascades and subsequent thrombus formation. This assumption is supported by histologic studies of resected specimens that

reveal organized thrombus within the lumen in association with intimal hyperplasia and inflammatory infiltration of the vessel wall. Ischemic effects of the disruption in flow may be magnified by embolization to the distal vasculature or vasospasm related to an increase in sympathetic tone [26,27].

Patients with ulnar artery thrombosis primarily present with complaints of pain, cold intolerance, and numbness. Pain is generally localized to the ulnar aspect of the hand and may be associated with a tender mass. Pain may be exacerbated by repetitive activity. Night pain and pain at rest can occur; however, this is unusual in the absence of significant ischemia.

The ulnar artery is intimately associated with the ulnar nerve in Guyon's canal. It is therefore not surprising that a significant number of patients will present with subjective complaints of numbness in the ulnar nerve distribution. Objective testing may be normal, however, subjective decreases in sensation and alteration in sweating in the ulnar nerve distribution have been reported in as many as 30% of patients [26].

Discomfort associated with exposure to cold is quite common. Symptoms usually involve the ulnar two or three digits and often follow a classic Raynaud's pattern. In patients with unilateral symptoms of Raynaud's, ulnar artery thrombosis should be strongly suspected and ruled out. Cold stress testing has confirmed alterations in blood flow and a characteristic digital temperature response has been described [26,31].

The treating physician should be suspicious of the diagnosis given the presenting symptoms previously discussed, and a careful evaluation of the ulnar artery should be conducted to evaluate its patency. The ulnar palm should be carefully inspected for any masses or tenderness overlying Guyon's canal, and a careful objective examination of the sensory and motor components of the ulnar nerve should be conducted The digits should be evaluated for ulcerations and trophic changes associated with chronic ischemia, and the nailbeds should be inspected for subungal splinter hemorrhages.

The diagnosis of ulnar artery thrombosis is made by the Allen test. Failure of the hand to reperfuse or absence of a dopplerable ulnar pulse with radial artery occlusion is diagnostic of ulnar artery occlusion. The exact location and extent of the thrombosis can be accurately evaluated by Doppler evaluation of the radial and ulnar arteries and the superficial arch. Digital plethysmography has also been found to be useful in the evaluation of these patients. By recording the flow response to sequential radial and ulnar artery occlusion, this device provides a quantitative digital Allen's test [26].

Arteriography, although not necessary for the diagnosis of ulnar artery thrombosis, provides invaluable information for formulating a course of treatment (Figure 2). The exact location and extent of the lesion, as well as the general health of the vessels and the amount of collateralization, can be accurately determined by arteriography. Although it is invasive, the authors of this review feel that the value of the information it provides far outweighs the risk and strongly recommend its use in the evaluation of this disorder.

E Figure 2

Fig. 2. Course of the ulnar artery and angiography showing ulnar artery thrombosis.

The management of ulnar artery thrombosis is controversial [7,18,26,27,31,37]. Treatments have included pharmacologic management, sympathectomy, thrombectomy, and arterial reconstruction. Recently, there has been a trend toward surgical management. An arteriogram is useful for preoperative planning in the surgical candidate. The exact location of the lesion as well as the extent of intimal injury and associated atherosclerotic disease can be assessed. If the thrombus is acute or the patient is a poor surgical candidate, an attempt can be made to treat with intra-arterial thrombolytics. However, if the lesion is chronic or there is significant intimal injury, it is unlikely that non-surgical management will be of significant long-term benefit.

Surgical approach is through Guyon's canal using a zigzag palmar incision. The ulnar nerve is decompressed and protected, and the thrombosed segment of artery is identified and resected to normal intima. In the presence of a complete arch, where there is strong bleeding from the distal aspect of the ulnar artery, simple ligation of the stumps has been recommended [31]. This eliminates the thrombus as a source of emboli. If the patient had clinical signs of significant ischemia preoperatively or there is inadequate backflow through the arch, arterial reconstruction is mandatory. This is usually best accomplished with the use of a reverse interposition vein graft harvested from the forearm.

Post-operatively the patient should be placed in a bulky hand dressing and a short-term course of an anti-thrombotic agent should be administered. A high incidence of recurrent thrombosis has been reported but does not require surgical exploration unless there is clinical evidence of significant ischemia. Results are usually very good with the majority of patients noting a significant improvement in symptoms [25,26,31]. Residual or recurrent symptoms can often be managed conservatively by cessation of smoking, biofeedback and

relaxation techniques, environmental modifications, and sympathetic blocks or sympathectomy.

#### Vasospastic disease

Episodic vasospasm of the digital arteries was described by Maurice Raynaud in 1862 and is referred to as Raynaud's Disease [3]. The disorder is characterized by a progression of ischemic symptoms initiated by stress or cold exposure. An initial period of blanching due to vasospasm is followed by a period of cyanosis. With re-warming there is reactive hyperemia leading to flushing of the skin in the digits which is often accompanied by pain and dysesthesias. Although the "white, blue, and red" is considered to be the classic presentation, patients with this disorder may experience a spectrum of symptoms with or without color change.

Episodic digital ischemia has been associated with a number of underlying organic disorders and environmental factors. When an associated disorder or factor is identified, the phenomenon is referred to as Raynaud's Syndrome. In 1932 Allen and Brown published the criteria that must be met to establish the diagnosis of Raynaud's Disease [3]. These include: (1) intermittent attacks of discoloration of the acral parts, (2) symmetrical or bilateral involvement, (3) absence of occlusion of peripheral arteries, (4) gangrene or atrophic changes, when present, are limited to the distal digital skin, (5) symptoms present for a minimum of 2 years, (6) absence of previous disease to which the abnormal vascular reactivity may be attributed, and (7) a predominance in women.

The etiology of Raynaud's phenomenon is unclear. It is associated with a number of disorders and factors including collagen vascular diseases, autoimmune disorders, hematologic and occlusive diseases, and occupational exposure to cold and vibration. This association would seem to be related to the fact that the vast majority of these conditions produce a spastic or obstructive arterial phenomenon. The pathophysiology of the disorder is believed to be secondary to several mechanisms [29]. Pathologic changes within the vasculature can lead to narrowing of the vessel lumens. These structural changes can also act to diminish the vessel's intrinsic ability to remain patent. Abnormalities in vasomotor tone can result in vasospasm and interruption of flow, and alterations in blood viscosity can lead to diminished flow. These mechanisms, occurring alone or in combination, result in the characteristic pattern of ischemia.

The diagnosis of Raynaud's is usually suggested by the history. Patients typically present with the classical pattern of symptoms preceded by stress or cold exposure. Non-invasive studies such as dopplers, plethysmography, and temperature monitoring are valuable tools in the evaluation of these patients, especially when combined with cold stress testing. Patients with Raynaud's have been shown to exhibit lower resting temperatures and digital flow than normals, experience more significant decreases in temperature with cooling, and take significantly longer to return to baseline with re-warming [36].

Arteriography plays a less prominent role in the evaluation of a patient with Raynaud's. It is useful in delineating proximal vascular lesions and should be obtained in unilateral cases and cases with progression of ischemic changes despite maximal medical management. It has also been found to be useful in evaluating the response to vasodilators in vasospastic disease.

The diagnosis of Raynaud's phenomenon is less important than the differentiation between the disease and the syndrome. Every effort must be made to exclude an underlying disorder whose treatment may take precedence over the Raynaud's. Late presentation such as the fifth decade, male sex, unilateral disease, and systemic symptoms should alert the physician to the probability of an associated disorder. Additionally, patients should be carefully followed for several years as the Raynaud's may be the presenting symptom of an as yet subclinical disorder. Differentiation of these groups also has prognostic significance. Patients with underlying disorders tend to follow a more progressive course and are less responsive to therapy.

The initial treatment of patients with Raynaud's is conservative. Efforts should be directed at limiting environmental factors that may contribute to the symptoms. Patients should be instructed to avoid cold exposure and to dress warmly and keep the hands protected. They should be urged to stop smoking and to limit their intake of caffeine. Some authors have advocated the use of biofeedback [11]. This technique has patients attempt to gain voluntary control over the autonomic mechanisms contributing to vasospasm by monitoring digital bloodflow and temperature.

Pharmacologic therapy has been directed at the suspected underlying mechanisms of the disorder. Significant attention has been directed at blocking the sympathetic nerves which are believed to be responsible for the increase in vasomotor tone. Intra-arterial reserpine, which acts by depleting catecholamines, has had some reported success [16]; however, it has largely been abandoned due to the deleterious effects of repeated injections. Oral administration of sympatholytic agents has been largely unsuccessful because of the unpleasant side effects of therapeutic doses [29]. Calcium channel blockers, such as nifedipine, and angiotensin converting enzyme (ACE) inhibitors, such as captopril, have been utilized in an attempt to inhibit the smooth muscle of the vessel walls and thereby limit vasospasm. These agents currently offer the most promise in the pharmacologic management of this disorder [5,29]. Anti-coagulants have been advocated to prevent small vessel thrombosis. Aspirin and persantine have been utilized to inhibit platelet aggregation [29].

Great care should be taken in the management of skin lesions. Established principles of wound care should be strictly followed and aggravating environments and agents avoided. Some studies have reported healing of ulcers with topical wound care and antibiotics. With progressive necrosis, amputation is often necessary. Attempts should be made to preserve as much length as possible, but not at the expense of leaving non-viable tissue.

In patients with progressive pain and ischemic changes who are unresponsive to medical therapy, surgical intervention may be indicated. Historically, efforts had been directed at removing the sympathetic input to the extremity by cervicothoracic sympathectomy. This approach had only temporary success and has been abandoned.

Flatt [10] first reported a technique of distal digital sympathectomy, which has subsequently been modified by Wilgis [35]. This technique entails separating the proper digital artery from the common digital nerve and stripping the adventitia of the artery for a distance of 2 cm. This technique has met with some success in carefully selected patients. Only patients who demonstrate improvement in pulse volume recording and cold stress testing after local anesthetic block are candidates. As with other treatment modalities, patients with underlying collagen vascular disorders do not have as favorable a response. Jones believed this may be related to periarterial fibrosis, causing extrinsic compression of the vessels and contributing to vasospasm [21,22]. He recommended a more extensive approach to expose the entire superficial arch and release fibrous septa. Segmental occlusions could be bypassed and an aggressive adventitial stripping conducted distally to the digital vessels. With careful patient selection these techniques have met with good short term results; however, it must be remembered that the majority of these patients have underlying disorders. It is likely that with time the progression of these diseases will lead to further small vessel disease and a recurrence of symptoms. These patients must therefore be followed over an extended period. Regular periodic examinations are necessary to identify changes early, maximize therapy, and prevent complications.

Vascular disorders of the upper extremity present a complex and challenging problem to the treating physician. The presentation is often subtle and the consequences of misdiagnosis or mistreatment severe. A thoughtful and thorough approach combining the history, physical exam, and appropriate diagnostic aids will provide the physician and patient with the greatest opportunity for a satisfactory outcome.

## References

1.

- 2. Aftaboddin M, Islam N, Jafa MAHM, et al: Management of isolated radial or ulnar arteries at the forearm. *J Traum* 38:149--151, 1995.
- 3. Allen EV: Thromboangitis Obliterans. Methods of diagnosis of chronic occlusive arterial lesions distal to the wrist with illustrative cases. *Am J Med Sci* 178:237, 1929.
- 4. Allen EV and Brown GE. Raynaud's Disease: A clinical study of 147 cases. *JAMA* 99:1472--1478, 1932.
- 5. Buehner JW and Koontz CL: The examination in the vascular laboratory. *Hand Clin*. 9:5--13, 1993.
- 6. Coffman JD: Calcium slow channel antagonists and Raynaud's phenomenon. *Intern Med* 5:107, 1984.
- 7. Coleman SS and Anson BJ: Arterial patterns in the hand based upon a study of 650 specimens. *Surg Gynecol Obstet* 113:409--424, 1961.
- 8. Conn J, Bergan JJ, Bell JI: Hypothenar hammer syndrome: Posttraumatic digital ischemia. *Surgery* 68:1122--1128, 1970.
- 9. Early MJ: The arterial supply of the thumb, first web, and index finger and its surgical application. *J Hand Surg* 11B:163--174, 1986.
- 10. Fitridge RA, Raptis S, Miller JH, et al: Upper extremity arterial injuries: Experience at the Royal Adelaide Hospital, 1969--1991. *J Vasc Surg*. 20:941--946, 1994.
- 11. Flatt AE: Digital artery sympathectomy. J Hand Surg 1980;5:550.
- 12. Freedman RR: Physiological mechanisms biofeedback. *Biofeedback Self-Regulation* 16:95, 1991.
- 13. Gelberman RH and Blasingame JP: The timed Allen test. *J Trauma* 21:477--479, 1981.
- 14. Gelberman RH, Blasingame JP, Fronek A, Dimick MP. Forearm arterial injuries. *J Hand Surg* 4:401--408, 1979.
- 15. Gelberman RH, Nunley JA, Koman LA, et al: The results of radial and ulnar arterial repair in the forearm. Experience in three medical centers. *J Bone Jt Surg* 64A:383--387, 1982.
- 16. Holder LE, Merine DS, Yang A: Nuclear medicine, contrast angiography, and magnetic resonance imaging for evaluation of vascular problems in the hand hand. *Hand Clin.* 9:85--114, 1993.
- 17. Hurst LN, Evans HB, Braun DH: Vasospasm control by intra-arterial reserpine. *Plastic Reconst Surg* 70:595, 1982.
- 18. Hutchinson DT: Color duplex imaging: Applications to upper extremity and microvascular surgery. *Hand Clin* 9:47--58, 1993.
- 19. Jelalian C, Mehrhof A, Cohen IK, et al: Streptokinase in the treatment of acute arterial occlusion of the hand. *J Hand Surg* 10A:534--538, 1985.
- 20. Johnson M, Ford M, Johansen K: Radial or ulnar artery laceration: Repair or ligate? *Arch Surg* 128:971--973, 1993.
- 21. Jones NF: Acute and chronic ischemia of the hand: Pathophysiology, treatment, and prognosis. *J Hand Surg* 16A:1074--1083, 1991.

- 22. Jones NF: Ischemia of the hand in ischemic disease: the potential role of microsurgical revasculariation and digital sympathectomy. *Clin Plast Surg* 16:547--556, 1989.
- 23. Jones NF: Ischemia of the hand in systemic disease. *Clin Plast Surg* 16:547, 1989.
- 24. Kleinert JM, Fleming SG, Abel CS, Firrell J. Radial and ulnar artery dominance in normal digits. *J Hand Surg* 14A:504--507, 1989.
- 25. Koman LA, Nunley JA, Goldner JL, et al: Isolated cold stress testing in the assessment of symptoms in the upper extremity: Preliminary communication. *J Hand Surg* 9A:305, 1984.
- 26. Koman LA, Smith BP, Smith TL: Stress testing in the evaluation of upper extremity perfusion. *Hand Clin* 9:59--83, 1993.
- 27. Koman LA and Urbaniak JR: Ulnar artery insufficiency: A guide to treatment. *J Hand Surg* 6:16--24, 1981.
- 28. Koman LA and Urbaniak JR: Ulnar artery thrombosis. *Hand Clin* 1:311--325, 1985.
- 29. LeClerq DC, Carlier AJ, Khuc T, et al: Improvement in the results in 64 ulnar sections associated with arterial repair. *J Hand Surg* 10:997--999, 1985.
- 30. Miller LM and Morgan RF. Vasospastic disorders. Etiology, recognition, and treatment. *Hand Clin* 9:171--187, 1993.
- 31. Nunley JA, Goldner RD, Koman A, et al: Arterial stump pressure: A determinant of arterial patency? *J Hand Surg* 12A:245--249, 1987.
- 32. Peris MD and Tomaino MM: Ulnar artery thrombosis: Evaluation and indications for operative treatment and surgical technique. *Am J Orth* 26:685--689, 1996.
- 33. Perry MO, Thal ER, Shires GT: Management of arterial injuries. *Ann Surg* 173:403--408, 1971.
- 34. Sumner DS: Noninvasive assessment of upper extremity and hand ischemia. *J Vasc Surg* 3:560--564, 1986.
- 35. Van Rosen S: Ein fall von thrombose in der arteria ulnaris nach einwirkung von stumpfer gewalt. *Acta Chir Scand* 73:500, 1934.
- 36. Wilgis EFS: Digital sympathectomy for vascular insufficiency. *Hand Clin* 1:361, 1985.
- 37. Wilgis EFS: Evaluation and treatment of chronic digital ischemia. *Ann Surg* 193:693--698, 1981.
- 38. Zimmerman N: Occlusive vascular disorders of the upper extremity. *Hand Clin* 9:139--150, 1993.